PROCEEDINGS OF THE CARDIAC SOCIETY OF
GREAT BRITAIN AND IRELAND

The NINTH ANNUAL GENERAL MEETING of the Cardiac Society of Great Britain and Ireland was held at The Royal Society of Medicine, London, on Thursday, April 12, 1945, under the Chairmanship of DONALD HALL.

The Chairman took the chair at 10.0 a.m.; 41 members and 22 temporary members and visitors were present.

The Chairman spoke of the loss that the Society and British Medicine had sustained in the death of Sir Thomas Lewis, F.R.S., who had accepted the Chairmanship for this meeting, and also by the death of W. T. Ritchie, the first Treasurer of the Society (1922–1931). He also welcomed the Canadian and American visitors.

An obituary notice of William Ritchie is on page 207: one of Sir Thomas Lewis will be in the January number.

PRIVATE BUSINESS

1. The minutes of the last meeting having been printed in the Journal (6, 247, 1944) were approved and signed.

2. The accounts, audited by East and Parsons-Smith, were approved: they showed a balance of £40 7s. 5d. The Council had decided that no subscription should be collected for the year 1945–46.

3. The Secretary, on the recommendation of the Council, was reappointed for another year, and William Evans was again asked to act as Assistant Secretary.

4. Three Ordinary Members were elected as Extra-Ordinary Members.

G. A. Allan Sir Maurice Cassidy A. M. Kennedy

5. The following new members were elected:—

Ordinary Members
R. Hilton
F. G. Hobson
T. Skene Keith
Bruce Maclean
B. E. Schlesinger

Associate Members
R. J. Duthie
F. M. Hilliard
J. G. M. Hamilton
E. Graham Jones
R. W. Snodgrass

Nine Associate Members were re-elected for another period of three years.

6. J. W. Brown, Grimsby, and H. Wallace-Jones, Liverpool, were elected members of the Council for the years 1945–49.

7. The Secretary reported that:

(1) He had received a letter from the Association of Physicians asking the Cardiac Society to consider the relationship between the Association and the various specialists' clubs and societies and that, with the approval of the Council, he had replied "that the general policy of the Cardiac Society had been to meet in the same town on the day before the Association and that they had no intention of changing this policy, though they wished to be free to make other arrangements from time to time, and that they would welcome a suggestion that at some meetings a session should be set aside as a joint meeting of the Association and the Cardiac Society and felt that this would be to the advantage of both bodies: at the same time the Council did not think it would be desirable for the Society to lose its identity or become a branch of a larger or wider Association."

(2) The Council had considered the question of Empire members and had decided to hold a special meeting to take further decisions in this matter. They wished the general approval of the Society for such a step and suggested that at the next Annual General Meeting they should bring forward detailed proposals of how this could best be carried out and any necessary changes in the rules of the Society. They thought that the question of a larger number of ordinary and associate members, with any necessary changes in the rules, should be considered at the same time. The Society gave general approval to some such development.
CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

SHORT COMMUNICATIONS

AURICULAR AND PERICARDIAL PRESSURES AND CARDIAC OUTPUT IN A CASE OF MALIGNANT PERICARDITIS

C. M. Fletcher (introduced)
(Published in full 1945, 7, 143)

THE CAROTID SHUDDER

David Lewes (introduced)
(Published in full 1945, 7, 171)

ELECTROCARDIOGRAMS OF A DYING HEART A FEW HOURS AFTER THE ONSET OF CORONARY THROMBOSIS

T. F. Cotton

EXPERIMENTAL AURICULAR FIBRILLATION

A. Morgan Jones and W. Schlapp (introduced)

The authors described experiments with the cat heart-lung preparation in which auricular fibrillation was induced and the cardiodynamics of the arrhythmia studied. A method of measuring the work capacity of the heart (cardiac reserve) was described and it was found that fibrillation reduces the cardiac reserve of the denervated cat heart by about one-third.

When fibrillation is induced at work rates which can be maintained during the arrhythmia, the heart output falls abruptly but is gradually restored to the original amount; the restoration of output is accompanied by a rise of auricular pressure. This period was termed adaptation. A period of stabilized fibrillation follows; during this the mean heart output and auricular pressure remain unchanged. When normal rhythm is restored the heart output rises above the original amount, the excess output quantitatively making up for the deficit acquired during the adaptive period. This period was termed recovery.

If the normal rhythm work rate at the onset of the arrhythmia exceeds the maximum work of which the heart is capable during fibrillation, the heart output is not maintained during fibrillation and the rate of work falls to the maximum fibrillating work rate. In these circumstances no stable state is achieved, for the auricular pressure continues to rise, the heart dilates beyond its optimal size and heart failure ensues. This outcome can be avoided only by reducing the work of the heart.

CARDIAC ENLARGEMENT WITH BRADYCARDIA IN RECRUITS

Crighton Bramwell

This communication concerned 39 cases that had been referred by medical boards of the Ministry of Labour and National Service on account of suspected cardiac enlargement for which there was no obvious cause. In 26 of these X-ray screening failed to confirm the presence of enlargement. Of the remaining 13, enlargement in 7 was trivial, and 4 of these were men of powerful physique, in whom slight enlargement was not unexpected. In 3 cases pathological cause for the enlargement was found, but the remaining 3 were healthy athletes, one a cross-country runner aged 34, the other two young men, aged 19 and 20, who participated actively in games and in both of whom the sitting pulse rate was 50.

The occurrence of cardiac enlargement in athletes who indulge in those forms of sport which necessitate prolonged and severe physical exertion is well known, but to what extent this enlargement is due to their training and to what extent it may be a congenital peculiarity it is not possible to say.

Comparative physiology shows that the heart ratio is high in athletic animals and low in sedentary animals, and there appears to be a general law that those animals which have a high heart ratio tend to have slow pulses with considerable vagal inhibition, while animals with a low heart ratio have rapid pulses over which the vagus exerts little influence. It is suggested that young healthy men with large hearts and slow pulses may be comparable to animals with a high heart ratio.
PROCEEDINGS OF THE CARDIAC SOCIETY OF

TWO CASES OF MALIGNANT HYPERTENSION TREATED BY NEPHRECTOMY

C. BRUCE PERRY

(Published in full, 1945, 7, 139)

2 : 1 HEART BLOCK IN PAROXYSMAL TACHYCARDIA

MAURICE CAMPBELL

(Published in full, 1945, 7, 183)

HEART FAILURE IN GENERALIZED PAGET'S DISEASE

O. G. EDHOLM (introduced), S. HOWARTH (introduced) and J. McMichael

A case of Paget’s disease with oedema and venous congestion was found to have a cardiac output of over 13 litres a minute. By plethysmography it was shown that the blood flow through the affected humerus was increased to about 20 times the flow through a normal bone. With extensive skeletal involvement the skeletal blood flow may be increased from a normal value of the order of 100 c.c. a minute to 3–4 litres a minute. This is responsible for the resulting hyperkinetic state of the circulation, with a collapsing pulse and manifestations of “cardiac failure.” (Clin. Sci., 1944, 5, in press).

THE EFFECT OF NITRITE ON THE INVERTED T WAVE

WILFRED STOKES (introduced)

(To be published in full)

DISCUSSION ON PATENT DUCTUS ARTERIOSUS AND ITS SURGICAL TREATMENT

The contributions of A. R. GILCHRIST (1945, 7, 1), GEOFFRY BOURNE (1945, 7, 91) and TERENCE EAST (1945, 7, 95) have been published in full.

JOHN HUNTER (introduced) spoke of the surgical methods he had used in ligaturing the patent ductus and of some of the difficulties. He emphasized the striking nature of the thrill felt even when it was not obvious on ordinary clinical examination.

J. W. BROWN said his experience showed that the outlook in patent ductus arteriosus was not altogether unsatisfactory without surgery. His remarks were based on 63 cases of patent ductus with a “machinery” murmur ranging in age from 3 to 45 years. There were 20 males and 43 females, and the periods of observation were for 16 cases, 5 years or less; 18 cases, 6–10 years; 29 cases, 11 or more years. Fourteen cases were below the average in development. The electrocardiogram showed a normal axis in 46; left axis in 9; and a right axis in 1. Two cases have died of bacterial endocarditis at the ages of 12 and 15. Regression of physical signs occurred in 2 cases. A slide was also shown of patent ductus in identical twins where the shunts were of different sizes, the larger shunt being accompanied by under-weight and stature in comparison with the smaller shunt. It was felt that there may be some indication for surgery in cases that are undersized, and who fail to develop normally, but undoubtedly many cases tolerated their lesion well, and in the above series there had been 8 pregnancies and no example of congestive failure.

DISEASE OF THE PERICARDIUM

TERENCE EAST opened the discussion.

In dry pericarditis the friction sounds are diagnostic, but sometimes the diagnosis from adjacent pleural friction is very difficult. It is curious how extensive pericarditis may be found at autopsy without friction sounds being audible during life. The cause of dry pericarditis is sometimes quite obscure. Such is the terminal pericarditis of renal disease, which carries such a bad prognosis. Instances of obscure origin, which clear up satisfactorily, are sometimes met with in adults. Some of these seem to develop a constricted sac later on. In children the pericarditis of acute rheumatism is part of a severe carditis. The enlargement of the heart which is usually present to considerable degree may simulate an effusion, but a large accumulation of fluid in these cases is not commonly seen. The signs of consolidation at the bases of the lungs, primarily and principally on the left side as a rule, are interesting.
There is no doubt that a rheumatic pneumonia is a cause in some cases, but in others a massive collapse also plays a part. This may be seen in adults. It is shown by the obvious restriction of expansion of the lower ribs, and the inhibition of the descent of the diaphragm on inspiration; serious embarrassment in breathing may result. It is therefore advisable to nurse these cases sitting up, instead of making them lie flat. The disappearance of much of the signs when the patient is propped up indicates the part collapse plays in causing the changes at the base of the left lung. When an effusion of any size is present in the sac it compresses the left lower lobe and causes collapse. In these circumstances it may be difficult to decide how large the pericardial effusion may be. It is probable that the sac must contain about 500 c.c. before definite signs are given. An effusion stretches the sac, and if it forms gradually a large volume can be accommodated without hindering the entry of blood into the heart. Rapid accumulation, as in hæmopericardium from rupture of the heart or aorta, causes acute tamponade, and at autopsy the heart is found to be contracted and empty. A very large effusion will also cause tamponade.

The same results are very gradually achieved in Pick’s syndrome from a constricted sac. The obstruction to the inflow leads to a rise in venous pressure and consequent engorgement of the jugular veins and liver. At the same time the rate of the heart rises, the output falls and the blood pressure sinks: The pulse may be paradoxical. Pressure on the lungs causes dyspncea. Cyanosis is common. These signs are usually an indication for paracentesis of the sac, which may be a useful and even a life-saving procedure. The site of election depends on the size of the effusion, and perhaps on its distribution, for sometimes it is loculated. The diagnosis may be helped by X-ray examination, particularly by screening, if this can be done. The diminished pulsation and slight change in shape on deep inspiration is typical, as well as the globular contour, obscuring the usual curves. The skiagram of a much dilated heart may give a very similar outline. In a simple serious effusion the cardiogram shows no characteristic changes. These depend on the underlying myocardium and are conspicuous in pyo-pericardium; concordant elevation of the R–T phase, followed by negative T waves later, appears in all limb leads, and are most conspicuous in lead II. R waves are not seen.

The diagnosis of the shrunken sac, of chronic constrictive pericarditis, is now readily made, and its relief can provide one of the most dramatic effects in surgery. But there is a fairly high proportion of failures, perhaps about 50 per cent. Examination of the sac in some cases shows that it is so thick and hard that relief is hopeless. The shadow of a calcified ring well inside the heart shadow may indicate the thickness of the sac. More information is needed on the failures. How far may isolated bands cause trouble? To what extent does removal of the front of the sac bring relief?

Although it is generally held that the heart is small in constrictive pericarditis, quite a proportion of patients have large heart shadows, perhaps about half. The low voltage curve and flat T waves are fairly constant. As to the cause of the condition, little is known. Tuberculosis may be responsible for some, probably less than was thought at first. Most seem to be completely obscure. Tuberculous pericarditis usually carries a bad prognosis. The disease reaches the sac from the mediastinal glands. Sometimes large effusions develop and need paracentesis. Sometimes the effusion is hemorrhagic and resembles that found with malignant disease inside the sac. It may be worth while to introduce air when the effusion is large, in the hope of preventing recurrent accumulation or organization of oedema. This may occur in any case and occasionally a constricted sac results; the wall is usually very thick.

Exo-pericardial adhesions are difficult to diagnose. An obliterated sac, unless it is constricted certainly gives no signs. A large right ventricle thrusting forwards may cause a typical retraction of the left lower ribs behind. There is a good deal of doubt how much these adhesions around the sac really matter. It seems very likely that the enlargement of the heart formerly ascribed to them is really due to the accompanying valvular lesions. Sometimes there is a drag on the costal cartilages in the front of the chest, and this may cause discomfort, which resection may relieve.

C. BRUCE PERRY discussed rheumatic disease of the pericardium.

Pericarditis in acute rheumatism is essentially merely part of a pancarditis and it is therefore difficult and, I think, undesirable to consider it alone as a separate entity. Although the recognition of pericarditis in a case of acute rheumatism may have certain diagnostic and prognostic value, it has this value by its implication rather than on account of the pericarditis itself.

As far as the pathology goes the condition is essentially an acute inflammation of the
pericardium with a sero-fibrinous exudate. In severe cases there may be a great deal of hemorrhage. Histologically it is possible to recognize the essential features of the rheumatic lesion: the same sort of cells are seen as in the classical "Aschoff" nodule but are loosely packed together as opposed to the more or less compact nodule found in the myocardium. There is always a little increase of pericardial fluid but for practical purposes this never exceeds an ounce or two.

Clinically the picture presented by a child with rheumatic pericarditis is so characteristic that it can be recognized almost at a glance. The ashen-grey cyanotic pallor, the restlessness, and the restrained short dry cough need no description. However, there is little reason to attribute these symptoms to the pericarditis: rather they should be regarded as due to the severe pancarditis—especially the myocardial lesion—which nearly always accompanies pericarditis. And the same picture is seen with no clinical evidence of extensive pericarditis.

The actual physical signs are, of course, the typical friction rub but this may be very evanescent in rheumatic cases. Cases with pericarditis usually have a great increase in the area of cardiac dullness and the cardiac shadow radiologically. However this increase in size is in the vast majority of cases due to dilatation of the heart due to the associated myocarditis. Pericardial effusion of clinical importance practically never occurs in acute rheumatism. Carey Coombs said that he had never seen a case that needed paracentesis. From my much smaller experience I can only confirm this. I have seen cases in which a paracentesis was mistakenly attempted but in these blood was withdrawn through the needle and it was, I think, clear that a dilated heart had been aspirated and not the pericardium.

It is commonly taught that the development of pericarditis in a case of acute rheumatism is a late or even terminal event and has therefore an extremely grave prognostic significance. In the vast majority of cases this is true, but not always. This is shown by the case of a little girl, aged 5 years 8 months, who was admitted to hospital with acute otitis media. Some days later she developed pain in the right hip followed in two or three days by pain in the other hip and the left shoulder. The next day there was a loud pericardial friction not at all over the precordium. An electrocardiogram showed a typical picture (Fig. 1). She rapidly improved and when the rub had disappeared there was heard an apical systolic murmur. However, with prolonged rest she finally made a complete recovery and now there is no clinical abnormality in the heart. Thus, just as in many cases the brunt of the damage falls on the mitral valve, occasionally the pericardium may be most affected.

As for treatment this must be directed to the carditis and, of course, complete and absolute rest with skilled nursing is essential. I do not use salicylates in cases with severe carditis as I have seen no evidence that they affect the course and I feel that the possible development of salicylism and vomiting would be fraught with such great danger that it is better avoided.

Fig. 1.—Electrocardiogram from child with rheumatic pericarditis.
One drug, and as far as I know one drug alone, may be life saving and that is morphia. It should be given in doses adequate to keep the child quiet and repeated as often and for as long as necessary.

The end result of rheumatic pericarditis is, of course, pericardial adhesions and in severe cases there may be a complete fusion of the two layers of pericardium. However, it appears to be very rare for acute rheumatism to produce the picture of constrictive pericarditis. In addition to the fusion of the pericardium there may be adhesions between the pericardium and the surrounding mediastinum. It is probable that these mediastino-pericardial adhesions are responsible for the physical signs—Broadbent’s sign and the peri-apical retraction. In such cases the clinical picture is usually dominated by the valvular and myocardial damage and, as in the acute phase, the pericardial lesion is a relatively unimportant component of the whole picture—the pancarditis.

John Parkinson showed lantern slides and outlined the radiology of pericardial disease, first making reference to the para-apical triangle, cysts and acculations of pericardial fluid, and tumours. In pericardial effusion the shortened vascular pedicle is significant; the cardio-hepatic angle is preserved and not obliterated as used to be taught. A table was shown to facilitate the differential diagnosis of effusion from gross cardiac enlargement. Effusion occasionally complicates cardiac infarction, and it appears to be not uncommon in myxedema.

In constrictive pericarditis there is neither great cardiac enlargement nor any characteristic outline. Fixation may be shown by the heart failing to fall during inspiration. Calcification is a most valuable sign present in 25–50 per cent; it is to be distinguished from calcified wall thrombi and from calcified deposits in the myocardial wall especially in cardiac aneurysm.

Tudor Edwards (introduced) said that it was difficult to diagnose pericardial suppuration which had formed as a complication of pulmonary or pleural suppuration. In such cases the cardiac outline was obscured by a loss of translucency either in one or both lung fields. Pericardial friction might be present, but the more certain diagnosis rested with exploratory aspiration of the pericardial sac. In this event it was important not to soil the pleura and it was best to direct the needle upwards and backwards at the left xiphisterno-costal angle.

Tudor Edwards spoke at greater length about constrictive pericarditis. He said that when calcification was present it was distributed irregularly as plaques of variable size and situated between the two pericardial layers although infiltrating the heart muscle itself. It was common experience to find pockets of thick creamy material in relation to the calcified plaques, material which had always proved sterile on culture. The condition appeared to be due to tuberculosis and three out of his series of eighteen patients had died of this subsequent to the operation. It was the aim of the operation to remove a sufficient amount of the constricting scar tissue to allow free movement of the ventricles. Unless an adequate window was made through the entire thickness of the pericardium the result of the operation would be a disappointing one. In most cases the pericardium still shows the original layers and the removal of the outer layer does not effect cardiac release, and it was necessary to remove the deeper visceral layer and permit herniation of the heart over the whole area of the operative field. It was his practice to clear the front of both ventricles, and the lateral wall of the left ventricle as far as the left phrenic nerve, and to free the cardiac apex. When possible the auriculo-ventricular groove is also cleared, but the auricles and caval areas are left undisturbed for they present the greatest operative hazards and their clearance adds nothing to the value of the operation. The pericardium is removed over the left ventricle before the right ventricle, and cardiac irregularity during manipulation and dissection of the calcium plaques is lessened by bathing the exposed heart with novocain. The left pleura is frequently opened when stripping it off the pericardium but this is no great disadvantage. Improvement from the operation has not been invariable; in some it has been dramatic although seldom immediate, and it has been best in those cases where the condition has not been long-standing.

William Evans spoke on the electrocardiogram of pericardial disease. He said that much had been written about this, its similarity to the tracing in cardiac infarction and the ways it differed from it. Early on it had been shown that the cardiographic changes arise from injury to the myocardium and occur independently of variations in the pressure within the pericardial sac, and the foremost worker in this field was a member of the Society, Fitzgerald Peel of Glasgow.

Changes in the cardiogram of acute pericarditis are confined to the R–T segment in the
early stages. Like that of cardiac infarction they are short-lived and are soon followed by inversion of the T wave, but unlike that of cardiac infarction the R–T deviation differs in its direction and in its form. Thus, the reciprocal relationship of the changes in leads I and III in infarction, namely, elevation of the R–T segment in one lead and depression in another, is absent in pericarditis; in the former the curve is said to be discordant and in the latter, concordant. Again, the elevated R–T curve of cardiac infarction has its convexity upwards and shows a coving effect, while in pericarditis the curve is saddle-like because the concavity is directed upwards. While the duration of the R–T changes is no longer than a few days, the deformity of the T wave which follows will last or disappear according to the progress of the myocardial injury, and it does not depend on the presence or absence of pericardial fluid. Thus a cardiogram in acute pericarditis might return to its normal state when the effusion disappears; sometimes the tracing hardly departs from the normal in the presence of a large pericardial effusion, while gross changes may take place in the absence of effusion.

Changes in the cardiogram of constrictive pericarditis are most prominent in the T wave although they are not confined to this. Their location in particular leads depends on the distribution of the myocardial injury from the pericardial disease, and on the degree of right heart preponderance. The changes are permanent and are not modified materially by operation. The voltage of the tracing is low in about one-half the cases, but by itself is not of great use in the differential diagnosis. Auricular fibrillation is not uncommon even in subjects under 30. Right axis deviation is often present. The P wave is usually broad and especially in lead II, and its ascending limb commonly exhibits a step or shelf. The Q wave, very often absent in all leads, is never prominent. R–T depression or elevation are uncommon features. The T wave changes are conveniently considered in two groups. In the first type there is inversion of T III and T II, and of T in CR3 and IVR, but not in CR7. Some of such changes may be attributed to right heart preponderance. In the second type there is inversion of T I and T II, and of T in IVR and CR7, while the T is low or inverted in CR1. Naturally a few cases show a combination of changes, but the cardiographic diagnosis of cardiac infarction and constrictive pericarditis can usually be told from an examination of the above characteristics. It is unsafe to rely on the concordant or discordant T wave in differential diagnosis because a concordant T might result from infarction of the anterior and posterior parts of the heart and simulate the findings in cardiac infarction complicated by pericarditis.